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The relationship between depolarization and the action potential complex of the cat's superior cervical ganglion.

By W. D. M. Paton and W. L. M. Perry. *National Institute for Medical Research, Mill Hill, London, N.W. 7*

Acetylcholine and nicotine produce ganglionic block accompanied by depolarization of the ganglion (Paton & Perry, 1951). These drugs also change the shape of the action potential excited preganglionically and recorded between the ganglion and the tied postganglionic trunk. This change is not caused by pentamethonium or D-tubocurarine, which produce block without depolarizing the ganglion. We have analysed these changes quantitatively, using non-polarizable electrodes and d.c. recording.

The slow waves of the normal ganglion action potential, described by Eccles (1935) as approximately exponential, can always be fitted graphically by a sum of two exponentially decaying waves, one negative and the other positive. Plotting the action potential logarithmically gives the time constant, t_p, of the slower positive wave directly; the time constant, t_n, of the quicker negative wave can then be obtained by subtraction. t_p and t_n were approximately 150 and 50 msec. respectively. Further, by extrapolation to zero time, estimates of a second pair of parameters, the initial magnitudes, N_o and P_o, of these waves can be made.

Although it has usually been assumed that the 'slow' waves and the initial 'spike' represent two distinct processes, we found that the difference between the initial magnitudes, N_o–P_o, is always approximately equal to the S_2 'spike' height of the normal action potential, and that if the two exponential waves are summed back to zero time, the curve obtained reproduces the entire action potential, apart from deviations due to the rising time of the 'spike' and to the S_3 and S_4 discharges.

Analysis of records obtained after drugs which depolarize the ganglion shows that the change in shape of the ganglion action potential is mainly attributable to a reduction of the parameter t_n to as little as 5 msec., whereas t_p is little altered. This great reduction in t_n is not found after drugs like pentamethonium.

In a normal ganglion the initial negativity, N_o, is usually about 3 times the 'spike' height. In a ganglion completely blocked by nicotine the maximum depolarization is of the same magnitude. Between these extremes, provided that it is possible to estimate N_o, the sum of N_o and the ganglion depolarization is also approximately constant at the same value. This suggests that the normal N_o represents the maximum depolarization of the normal cell membrane, and that after partial depolarization by nicotine or acetylcholine, N_o represents the

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remaining possible depolarization. On this view, therefore, the principal action of depolarizing drugs on the ganglion is, through the depolarization, to reduce the initial magnitude of the negative wave in response to preganglionic stimulation, and greatly to accelerate its decay.

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